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by

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It was in the paper reported by TAKAYASU (1908)[49] under the title of "A case of the curious changes in the central vessels of the retina" that this pulseless disease appeared for the first time in the medical literature in this country. As late as 1948, SHIMIZU and SANO[46] made a detailed report or the clinical pathologic examination of this disease, which came to be designated as "pulseless disease". Since then, the case report of this disease has rapidly increased in number. According to AIBA[2], there had been 218 cases reported in this country by January 1961. ROSS MCKUSICK (1953)[44] and ASK UPMARK et al. (1956)[5] regarded a series of conditions in which the pulsation of an artery arising from the aorta had been weakened or had disappeared, irrespective of its etiology, as "aortic arch syndrome", among which they included young female arteritis that seemed to correspond to the pulseless disease as well as syphilis, arteriosclerosis, traumas, congenital anomalies, aortic dissociation, thrombi, emboli, and mediastinal tumors. It is etiologically observed in our country that pulseless disease is related to tuberculosis (SHIMIZU and SANO[46], NASU[40]), while its relationship to rheumatism is noticed in the foreign countries (ASK UPMARK, 1934[4]). In any way, it is agreed that the essential aspect of this disease is regarded as arteritis; and the mechanism of the allergic-hyperergic reaction is inferred. Although various causes for this disease have been mentioned such as tuberculosis, streptococcal infections, allergic reactions, rheumatic diseases, endocrinological disorders and collagen diseases, there is an opinion that if this disease has a characteristic pathologic picture of non-specific arteritis, it is advisable to examine it from the stand-point of vasculitis as a clinical-pathologically independent entity, without having it comprehended into a large category of aortic arch syndrome[24].

In the foreign countries, there are a large number of cases reported concerning the surgical treatment of aortic arch syndrome or occlusive diseases of the carotid artery, such as by DE BAKEY[8-10], FREEMANN[13], BAHNSON[6] and others. Their operative results were very good. Similarly in our country, transplantation of synthetic vessels has been performed on pulseless disease, and fairly good results have been obtained. They are, however, considerably inferior to those results obtained by DE BAKEY and others. This is naturally expected from the fact that the predominant pathological changes of pulseless disease appear in the tunica media of the main arteries and accompany acute inflammatory pictures over the whole layer of the vascular wall[39][40][46], covering the wide scope, while

the pathological changes of the cases reported in the foreign countries consist mainly of degeneration of the tunica interna due to atherosclerosis[11)16)] and do not include inflammatory changes. It is, therefore, necessary to reconsider its surgical therapy from the different point of view.

Case 1. a 42-year-old female.

Chief complaints: Blacking out and hardness of hearing

Present illness: First childbirth was at the age of 33. About 6 months later, she got blacking out when she looked up in order to hang out the washing to dry. Since then, she came to suffer from vertigo and blacking out even while walking. These disappeared after she looked down for a while, but occurred again when she looked up. This made her walk downcast with her head down.

Six years before, she suffered from ringing and hardness of hearing in the left ear, and was given a diagnosis of left otitis media.

Three years before, she felt as if the heart were about to stop, and suffered from continuous pyrexia (38.5°C) of unknown cause for about 1 week. As she subsequently continued to be annoyed by flush in the face, a sense of heaviness in the occiput, stiffness of both shoulders and anorexia, she was admitted to the department of medicine in this hospital on July 21, 1963. Both the radial arteries were hard to be palpated, and hypotension at both the arms were pointed out. She was discharged from the hospital in September 1963, while her condition had been still hanging in the balance.

In addition, she had been suddenly attacked by hardness of hearing in the right ear 3 years before, which was cured in about 1 month. Recently, she complained again of hardness of hearing in the right ear which became severer since these 1 to 2 months. On the other hand, anorexia and vomiting after meals turned serious. She was again hospitalized in the department of medicine on May 29, 1964; and referred to the department of surgery for pulseless disease in September 1964.

Status praesens: Her constitution is moderate, but she is slightly slender, taking a peculiar poise of drooping the head. The complexion is a little purple red, and vascular dilation is prominent in the inner canthus. No perforation in the nasal septum, but the denture is extremely in disorder. No gross abnormalities in the cervical region. Both the carotid arteries are not palpable at all. No systolic murmur is audible. The carotid arterial reflex is not remarkable. No physical abnormalities noted in the heart and lung. Pulmonary marking is mildly intensified on the x-ray pictures. No noticeable changes observed on the ECG finding. The extremities show no paresthesia, dyskinesia and abnormal reflex. Both the arms are apparently a little slim giving an impression on having been abruptly thinned at the phalangeal regions. Any of the pulsation of subclavian, axillary, brachial and radial arteries can not be palpated. The arteries of the lower extremities are well palpated. The blood pressure of the popliteal artery is 144/88 mmHg on the right and 140/80 mmHg on the left.

Laboratory findings: RBC 3,930,000. Sahli 84%. WBC 6,000. Blood platelet 227,940. Bleeding time 1 minute. Coagulation time (SAHLI-FONIO) starting at 9 minutes and closing at 13 minutes. Prothrombin time 25 seconds (using Indion). Total serum protein 7.0 g/dl. Protein fractions: albumin 44.8%, α_1-globulin 1.2%, α_2-globulin 12.3%, β-

globulin 21.1%. γ-globulin 22.6%. Total cholesterol 230 mg/dl. Free cholesterol 60 mg/dl. Na 138 mEq/L. K 4.0 mEq/L. Liver bunction tests are all within normal limit. Erthrocyte sedimentation rate S_1 35 mm. Blood WA.'s reaction (−). MURATA's reaction (+). ASLO 166 TODD u. CRP (−). RAT(−). ROBINSON-POWER-KELPER water test (−). Tuberculin reaction (−). FISHBERG's concentration test normal. PSP normal.

Ocular findings: Visual power Rv=0.8 Lv=0.8 (n. c.). Mild concentric contraction of the visual field. Bulber conjunctiva and pupils are normal. Intraocular pressure is 19 mm. Hg on both sides. Eyeground blood pressure is lowered, 22 mmHg at maximum and 17 mm. Hg at minimum. Marked venous dilation and tortuosity and indistinct margins of the papilla in the eyegrounds are demonstrated.

Hearing test: Hearing in impaired on both bone and air conductions. The disturbance is particularly severe in the left ear. She uses a hearing aid but sometimes fails to understand ordinal conversation.

Circulatory findings: Venous pressure (left elbow) vein 47 mmH_2O (MORITZ-TOBURA's modified method). Circulation time 20 seconds (Decholin), 7 seconds (Alinamin). In the other words, the arm-lung time is normal, but the arm-tongue time is slightly delayed.

X-ray findings: Retrograde aortography using a catheter revealed no brachiocephalic artery, common carotid arteries, subclavian arteries, nor vertebral arteries. Collateral routes were seen to be enlarged in the cervical and intercostal regions (Figure 1).

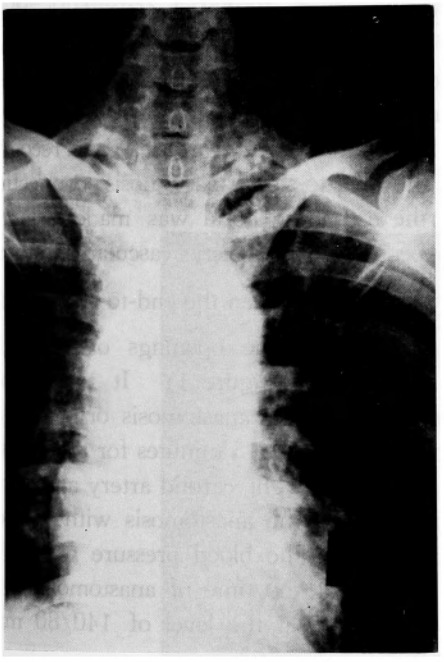

Fig. 1. Arteriogram of Case 1 showing ro visualization of the brachiocephalic, left and right common carotid, and left and right subclavian arteries. Marked hyperplasia of the collateral routes are seen bilaterally in the cervical portion.

Operation (performed on September 7, 1964). Skin incisions were made on the anterior margins of both the sternocleidomastoid muscles in the superior cervical regions under general GOF anesthesia at normal temperature. To begin with, both the superior cervical sympathetic ganglions were resected. The common carotid arteries, being adherent to the surrounding connective tissue on both sides, were completely obstructed and showed cord-like cicatrization. On the right side, the inner lumen peripheral to the point about 2 cm. from the remification on the heart side was well open, and the wall was not thickened. There was no remarkable inflammatory change observed and some degree of pulsation was felt. On the left side, the obstruction on the heart side was much closer in site to the ramification, and the pulse on the peripheral side was a little fainter. In the next place, the ascending aorta, aortic arch and its ramifications were exposed by vertical section of the sternal bone. There was not so marked change found in the wall of the

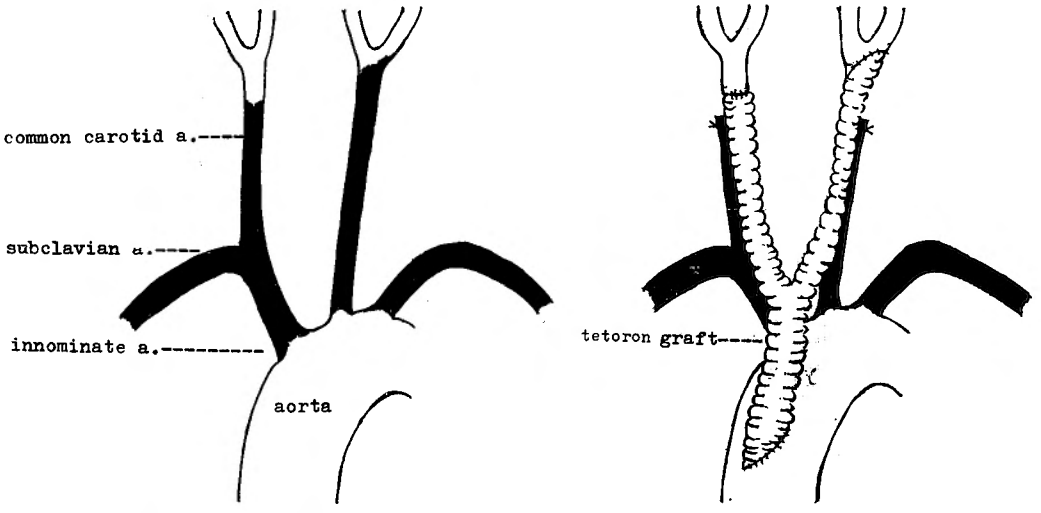

Fig. 2. Case 1. Operative technique and findings at operation (sites of obstruction are shown in black).

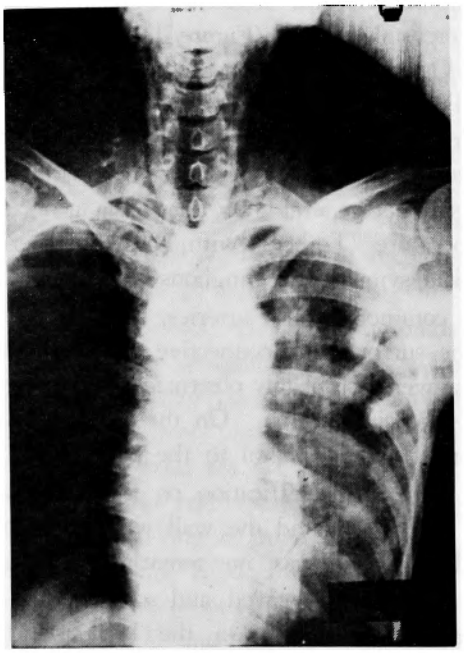

Fig. 3. Case 1. Arteriogram taken after the synthetic vasculer grafting. The Y-shaped synthetic vessel grafted in the ascending aorta is shown to be well patent.

aorta ; but the brachiocephalic artery, left common carotid artery and left subclavian artery were completely obstructed at their first portions and were adherent to the surrounding connective tissue in cord-like cicatrization. As a back flow from the patent right and left cranial carotid arteries was recognized, the end-to-side anastomosis connecting with the ascending aorta was made using a Y-shaped tetoron synthetic vascular prosthesis ($16 < \frac{8}{8}$mm), and then the end-to-end anastomosis to connect the openings of both the carotid arteries. (Figure 1) It took about 60 minute to make anastomosis on the wall of the aorta, about 15 minutes for the anastomosis with the right carotid artery and about 30 minutes for the anastomosis with the left carotid artery. The blood pressure was 160/110 mm.Hg at the time of anastomoses, but was maintained at the level of 140/80 mm. Hg after the operation.

As for the anticoagulant therapy, 20 mg of Heparin was administered into the Tetoron graft and 30 mg. of Heparin was injected systemically immediate after the preclotting following the anastomosis between the aortic wall and the tetoron graft. For 7 days following the operation, Heparin was continuously administered intravenously by drip method in dosage of 150 mg. daily, and thereafter Sintrom

was given for about 1 month in order to maintain the prothrombin time at about 30 seconds. Besides, streptokinase and chymotrypsin were employed in combination for about 40 days after 3 rd. postoperative day as the fibrinolystic agents.

As the anti-inflammatory agent against the arteritis, adrenocortical hormone was administered for about 2 weeks.

Postoperative course : Because of incomplete drainage, hematoma was formed causing the pressure symptoms of the heart such as dyspnea and hypotension. On the 2nd day, the operative wounds were reopened and the hematoma was evacuated. Following the removal, the blood pressure restored to normal, and the general condition was improved. In addition, no cerebral nervous symptoms due to the circulatory disturbance were left at all. The hearing disturbance in the right ear was so improved that the patient became able to enjoy conversation without using a hearing aid. The peculiar poise of drooping the head, which had been observed before the operation, was corrected. She became free from both vertigo and blacking out when she looked up. Dilation and tortuosity of the veins of eye-ground almost subsided and the color of purple red in the face disappeared. The angiogram taken 3 months after the operation showed that the transplanted vessel was well patent (Figure 3). This improved condition is now seen to have been maintained for 1.5 years following the operation.

Histological findings : The inner lumen of the common carotid artery was occluded by the organized thrombus, demonstrating almost no inflammation of active phase. The picture of vascularisation was observed among the prelifevated fibrous tissue. (Figure 4). The elastic and muscle fibers of the tunica media disappeared completely, and deposition of amorphous substances and fibrosis were demonstrated (Figure 5). In the adventitia, cicatrization was remarkable, and the inflammatory reactions chiefly of neutrophils and partially of lymphocytes remained in the surrounding of the nutrient vessels. The

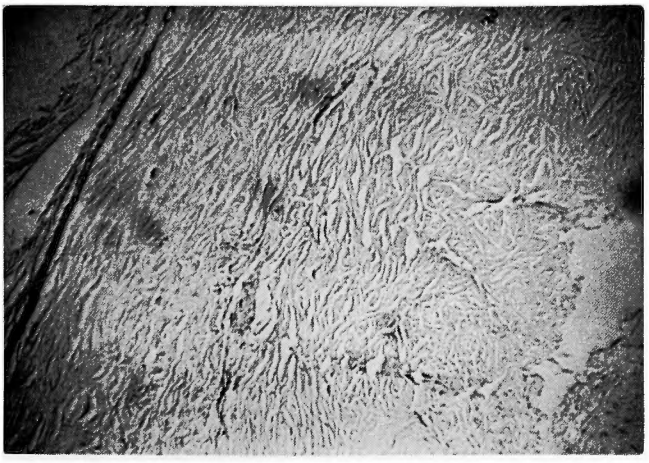

Fig. 4. Case 1. Common carotid artery. Its lumen is obstructed with an organized thrombus, and the muscle and elastic fibers in the tunica media have completely disappeared. Both the tunica media and the adventitia show marked cicatrization.

(H. E. staining)

adventitial area of the aorta had also the infiltration of neutrophils and lymphocytes surrounding the arterioles, but it was milder than in the carotid artery (Figure 6). In the tunica media, there was deposition of inhomogeneous substances, but the elastic fibers were well preserved (Figure 7). The tunica interna was coarse and proliferative, but showed no inflammatory cellular infiltration.

Case 2. a 25-year-old female

Chief complaints: Ringing in the left ear and attacks of syncope.

Present illness: Since May 1962, she had been continuously suffered from slight fever, general fatigue and pain in the right chest. She was admitted to a certain hospital, and was treated by SM, PAS and INHA. Thecondition was improved for a

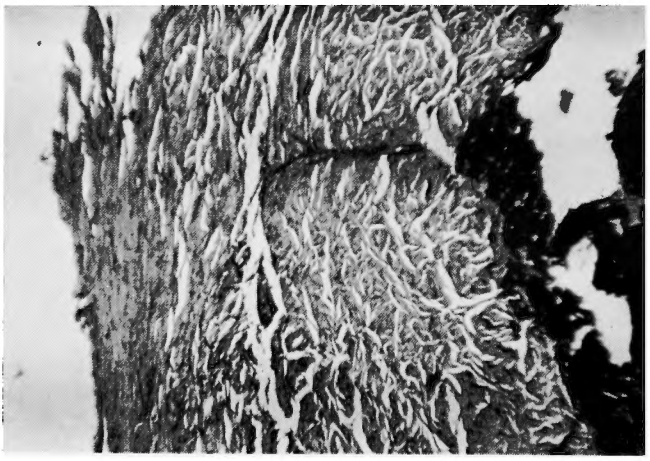

Fig. 5. Case 1. Common carotid artery. Both the elastic fibers of the tunica media and the lamina elastica interna have completely disappeared (Elastica- van Gieson staining).

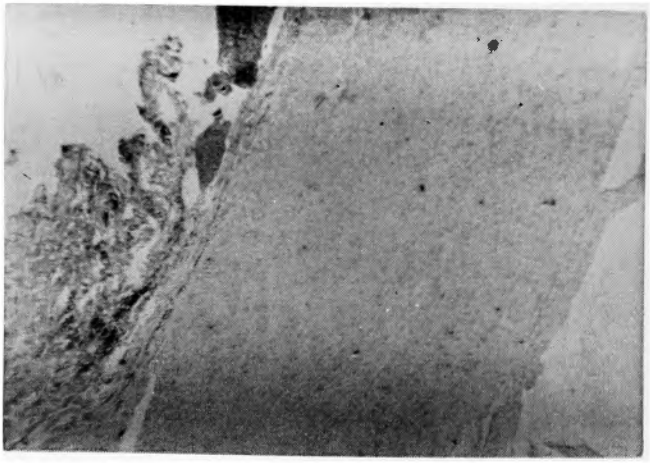

Fig. 6. Case 1. Aortic wall. A mild cell infiltration is seen in the surrounding of the nutrient vessels of the adventitia, and deposition of inhomogenous substances in the tunica media (H. E. staining).

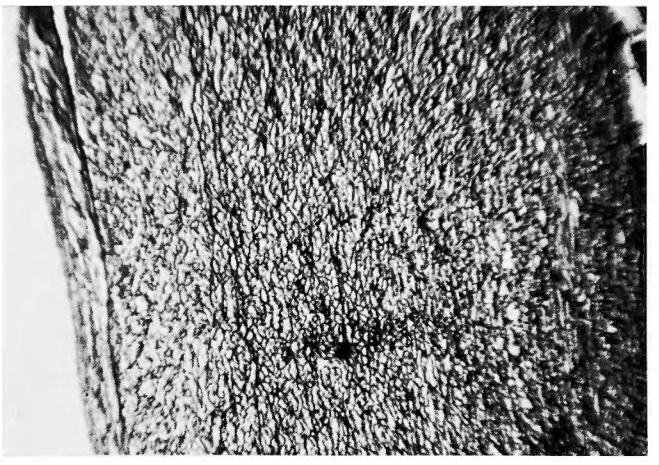

Fig. 7. Case 1. Aortic wall. Both the elastic fibers of the tunica media and the lamina elastica interna are well preserved (Elastica-van Gieson staining).

while ; but slight fever, pain in the right chest, nausea after meals, vomiting, tachycardia and palpitation recurred and resisted since the middle of July.

On August 30, she was discharged from the hospital and had since been under ambulatory treatment. The above symptoms were, however, further aggravated, and systemic convulsion of about 2 hours duration occurred following sudden nodding movement on September 24, and she was admitted again to the same hospital, but moved to another hospital on October 24 because of fever with unknown etiology and leucocytosis had still resisted. She had been treated as septicemia, but acne-like eruptions appeared in the face and the anterior thoracic area since November, and a diagnosis of collagen disease was given. The condition was improved by the treatment with Ilotycin and Predonin, and the patient was discharged from the hospital on October 28, 1963. One week later again the symptom of retching, fever, and leucocytosis (20,900) appeared. Since about that time, she began to feel ringing in the left ear, hardness of hearing and vertigo at standing.

On November 3, 1963, she was admitted at a certain hospital in Osaka. It was found accidentally by a nurse on December 25, 1963 that the left radial artery was hard to be palpated, and a diagnosis of pulseless disease was made. Subsequently, in February 1964, she suddenly felt vertigo and became quite black before her eyes when she was going to look at a coming shuttlecook while playing badminton.

She had been under ambulatory treatment since discharge from the hospital on March 29, 1964. The symptoms such as anorexia, general fatigue, retching, ringing in the left ear, vertigo, pain in the right chest and fever became aggravated since about July 1964. She was again hospitalized on August 2, 1964 because she came to feel difficulty in breathing due to the pain in the right chest and lost consciousness following systemic convulsion. She had the repeated attacks of syncope lasting from 3 hours to 1 day almost everyday up to August 15. The condition was relieved by administration of adrenocortical hormone (Paramesone), and she was referred to the department of surgery on September 24, 1964 for the surgical treatment of pulseless disease.

Status praesens: She is moderately built, but slightly emaciated. The complexion is a little pale, with acne-like eruptions observed on the face. No macroscopic changes in the cervical region. The left carotid artery is felt a little faint as compared with the right. The systolic purring thrill is felt over the entire area. Ringing in the left ear disappears by the compression of carotid artery. The carotid sinus reflex is not remarkable. On the other hand, systolic attrition murmur is heard at the supraclavicular fossa.

The heart sounds in the base are not clear at systole. Particularly systolic murmur is heard at aortic valve. Besides, a systolic attrition murmur is audible also in the right posterior lung field. No physical abnormality is found in the lung. The x-ray picture of the chest reveals the protrusion in the area from the aortic arch to the descending aorta. Sinus tachycardia is demonstrated on ECG. A phonocardiogram shows systolic low-pitched murmurs in the pulmonary and the aortic area. There is a pain on tapping on the 5 th thoracic vertebra.

There is no abnormality demonstrated in the abdomen.

Paresthesia, dyskinesia and abnormal reflexes are not seen in the extremities. Neither hypoplasia, edema nor anemia are demonstrated.

The right radial artery is well tonic, with the pulse being regular but too rapid (at the rate of 110 per minute). The blood pressure is 104/40 mm.Hg. On the other hand, left subclavian artery, left axillary artery and left radial artery can not be palpated, and therefore it is impossible to determine the blood pressure in this area. The pulse in the lower extremities is distinctly felt. The popliteal arterial blood pressure is 154/50 mm.Hg on the right and 160/40 mm.Hg on the left.

Laboratory findings: RBC 3,740,000 (irregular in size, reticulocyte 63%). Sahli 73%, Hematocrit 36%. WBC 10,400. Bloodplatelet 347,820. Bleeding time 1 minute. Coagulation time starting at 6 minutes and closing in 15 minutes. Prothrombin time 21 seconds. Total serum protein 6.0 g/dl. Protein fractions: albumin 49.7%, α-globulin 10.2%, β-globulin 12·3%, γ-globulin 27.8%. Total cholesterol 240 mg/dl. Free cholesterol 70 mg/dl. Serum Na 139 mEq/l. Serum K 4.0 mEq/l. NPN 29 mg/dl. Alkaline phosphatase 1.4 BODANSKY u. Liver functions tests are all within normal limit. GOT 24. GPT 18. Erythrocyte sedimentation rate S_1 126 mm. Wa's reaction uncertain. Tuberculine reaction positive. ASLO 333 TODD u. CRP(卅). RAT(卅). Blood culture yields no bacilli.

Ocular findings: Visual power Rv=0.7 (n. c.). There is a serious degree of concentric contraction of the visual field. Bulbar conjunctiva and pupils are normal. The ocular pressure is 17 mm.Hg on both sides. Eyeground blood pressure 78/28 mm.Hg arterial and less than 20 mm.Hg venous on the right, more than 130/20 mm.Hg arterial and less than 19 mm.Hg venous on the left.

Eyeground findings: There is a moderate papillar hyperemia on both sides, the margins being obscure. The arterial walls are thickened. The veins are strongly dilated and tortuous. No arteriovenous anastomosis is observed. In short, the findings seem to belong the Grade 1 (vascular dilation stage) classified by YANAGIDA[59].

Hearing test: Hearing of a high-pitched sound on air conduction is considerably disturbed on the left ear.

X-ray findings: Retrograde aortography using a catheter showed stenosis of the right

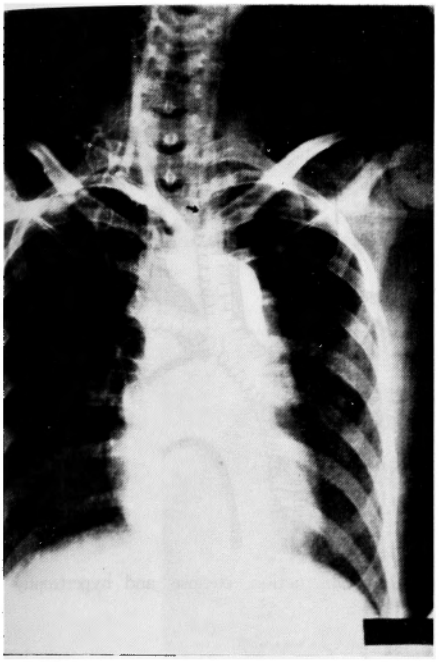

Fig. 8. Arteriogram of Case 2 showing stenosis of the left common carotid artery, and obstruction in the first portion of the right subclavian artery and in the left subclavian artery.

subclavian artery at its first portion, stenosis of the left common carotid artery at the portion from its origin to the ramification and complete obstruction in the portion from the origin of the left subclavian artery to the left axillary artery and complete obstruction of the left vertebral artery (Figure 8).

Operation : CRP was still strongly positive, but the white blood cell count turned normal. Thereupon, reconstruction of the blood circulation in the left carotid artery was performed on October 29, 1964 for the treatment of frequent attacks of syncope, vertigo, ringing of the left ear, hearing disturbance and advancing arterial stenosis. A skin incision was given on the anterior margin of the sternocleidomastoid muscle in the left upper cervical region, and the sympathetic ganglion in the left upper cervical region was first resected. The left common carotid artery was cicatrized over the whole length adhering to the surrounding connective tissues and gave an appearance of a hard cord ; but faint pulsation was felt and a slight blood stream was recognized. This cord-like thickening of the vascular wall extended as far as the ramification, but the internal and external carotid arteries were well patent, and the inflammatory thickening of the arterial wall was mild. The left subclavian artery was cicatrized like a cord as far as the transitional portion to the axillary artery, adhering to the surrounding connective tissue, and was completely obstructed. The portion peripheral to the axillary was, however, well open.

The ascending part of the thoracic aorta and aortic arch were exposed by the vertical section of the sternum. The aortic wall as well as the brachiocephalic artery showed redness and serious inflammatory hypertrophy of the wall, and considerably adhered to the surrounding connective tissues. A slight patency was demonstrated in the left common carotid artery, but the stenosis at its first portion was serious, with the left subclavian artery obstructed completely from its origin. Both the arteries were seen to be in the shape of a hard cord. Thereupon, the end-to-side anastomosis using a Y-shaped Tetoron vascularprosthesis ($14 < \frac{7}{7}$ mm) was first performed on the ascending aorta. The left external carotid artery was ligated and the remification was resected including the glomus caroticum, and then the end-to-end anastomosis was made with one branch connected to the internal carotid artery and the other branch to the left axillary (Figure 9). At the operation of anastomosis, the blood stream was blocked for about 57 minutes in the brachiocephalic artery and for about 15 minutes in the left carotid artery ; and the blood

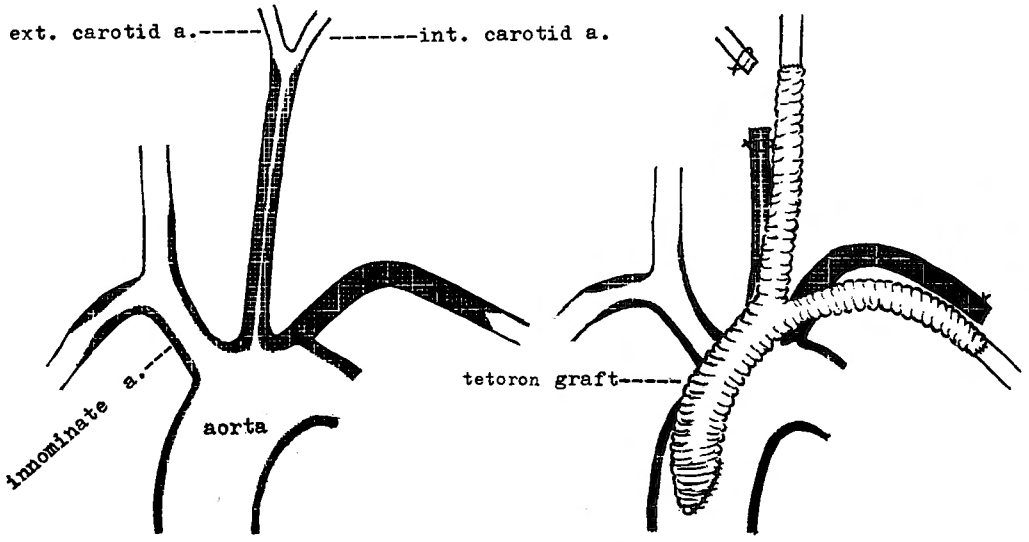

Fig. 9. Case 2. Operative technique and findings on operation (Obstruction, stenosis and hypertrophy of the arterial wall are shown in black).

pressure was maintained at the level of 190/70 mm.Hg.

As the anticoagulant therapy, 20 mg. of Heparin was injected into the Tetoron graft and 30 mg. systemically by divided injection immediately after the preclotting following the aortic wall anastomosis. In addition, Heparin was continuously administered by drip intravenous injection in dosage of 100 to 150 mg. daily for 9 days following the operation. On the other hand, adrenocortical hormone was simultaneously employed as an antiinflammatory agent.

Postoperative course: No cerebral symptoms due to the blockage of blood stream occurred after the operation. The arteries in the left arm and the left carotid artery became well palpable. Hardness of hearing and ringing in the left ear were improved. On the other hand, tachycardia and hypertension resisted but restored closely to normal since the 6th day following the operation. On ECG, a slight fall of ST_{123} was seen. Roentgenologically, the lung field was normal. The postoperative general condition seemed to be comparatively good, but the patient died on the 9th postoperative day from a sudden attack of hypotension and dyspnea. At autopsy, the transplanted synthetic vessel was seem to be patent with no thrombi, but the brachiocephalic artery and the starting area of the left common carotid artery were completely obstructed with newly formed thrombi. It was also found that the newly formed thrombi were adherent to the valves in both the ventricles of the heart. Besides, congestion was found in the lower lobes of the lungs, and a small quantity of exudate in the thoracic cavity.

Histological findings: The left common carotid artery showed the pictures of vasculitis over the whole layers; predominantly fibrosis in the tunica interna and adventitia, and serious infiltration of inflammatory cells (mainly of plasmocytes and lymphocytes, and a small quantity of acidophils) and granulation incorporating Langhans' giant cells in the tunca media (Figure 10). There was no fibrinoid degeneration observed. By Elastica

V. G. staining, it was shown that the elastica interna was maintained, but the break-down of the elastic fibers in the tunica media was serious. In the aortic wall, the tunica interna showed a serious degree of hypertrophy, but the tunica media was contrarily atrophied. and there was fibrosis of the adventitia with infiltration of lymphocytes and plasmocytes in the surrounding of the nutrient vessels of the adventitia (Figure 11). The elastic fibers were seen by Elastica V. G. staining to be broken into fragments. There was no contributory findings other than diastasis of the cardiac-muscle fibers and a slight fibrosis in the heart.

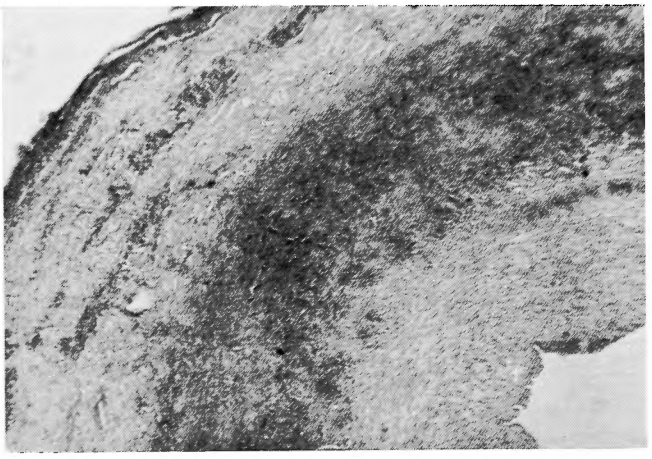

Fig. 10. Case 2. Left common carotid artery. Serious inflammatory cell infiltration and granulation incorporating giant cells are seen in the tunica media. Fibrosis and marked hypertrophy are seen in the tunica interna and the adventitia (H. E. staining).

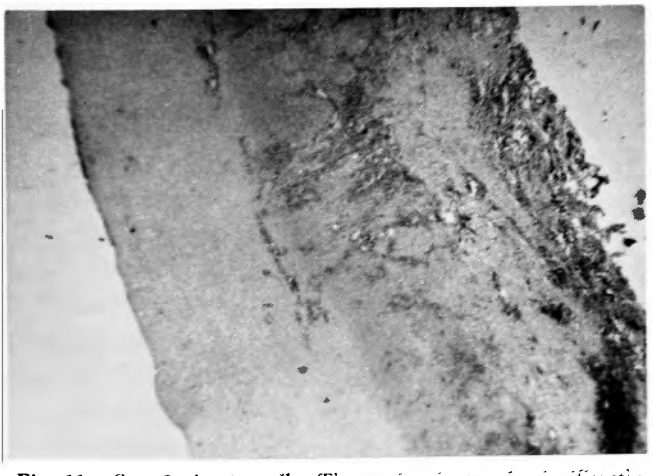

Fig. 11. Case 2. Aortic wall. The tunica interna is significantly hypertrophied, but the tunica media is rather atrophied. A considerable degree of infiltration of lymphocytes and plasmocytes is seen in the surrounding of the nutrient vessels of the adventitia (H. E. staining).

DISCUSSION

It has been reported that adrenocortical hormone[2)3)4)14)17)21)32)35)36)48)55)], anticoagulant agents[15)34)] and vasodilators[41)43)] produce fairly satisfactory results in the internal treatment of pulseless disease. However, these kinds of treatment seem to cause only a temporary arrest of progress in the condition, besides can be considered as being rather insufficient treatment for the cerebral circulatory disturbances and visual disorders due to obstruction of the carotid artery, and also for the attacks of syncope and vertigo due to the promotion of reflexes of the carotid artery.

Surgically, the treatment can be given from two directions; i. e. inhibition of the promoted reflexes of the carotid artery, and improvement of cerebral circulation. With respect to the former, resection of the sympathetic plexus of the carotid arterial sinus and the glomus caroticum has been performed[2)23)27)]. According to the statistic investigation made by AIBA of the Department of Neurosurgery in TOKYO University, the observation carried out 2.5 to 15 years following the operations showed that the bilateral resection was effective at the rate of 100% and the unilateral one effective at the rate of 66.7% on the symptoms of the promoted reflexes. However, in addition to the inhibition of the promoted reflexes of the carotid artery, there still remains a problem of cerebral circulatory disturbances.

According to DE BAKEY[9)], thromboendoarterectomy was reported as the satisfactory operation, i. e. vascular opening, in 34 out of 37 cases treated (91.9%), while AIBA reported that it produced only 8% improvement in the treatment of pulseless disease. DE BAKEY's cases were, as mentioned above, the degenerative pathological changes of the tunica interna due to atherosclerosis, and had no pictures of inflammation. On the other hand, pulseless disease appears as panvasculitis affecting the whole layers of the arterial wall, with the main pathological changes in the tunica media ; consequently, the inner lining of the vascular cavity remains pathologic even after the operation, and additionally the pathological changes of this disease is not segmental but extent from the aortic arch down to the rather peripheral areas of its main branching arteries. It is, therefore, indicated that application of this method to pulseless disease is unreasonable. Besides, there are reports concerning the autotransplantation of the vein and anastomosis of the internal and external carotid arteries. These methods are, however, limited in application in the same meaning as in thromboendoarterectomy and it can be hardly said that these methods produce good results.

Of the case reports concerning the synthetic vascular transplantation in pulseless disease in our country[2)18)25)26)28)30)31)50)51)52)53)54)], we reviewed the convincing cases of which the prognosis had been stated evidently as well as our cases reported here in this paper (Table 1). The results were fairly good, with vascular opening obtained in 13 out of the 15 cases reported (68.4%). This is, however, by far less excellent than 100% vascular opening obtained by DE BAKEY[9)] in 38 cases of the grafting. The prognosis was seen in 6 cases. Of them, 3 cases seemed due to thrombus formation, 2 cases died from hemorrhage, and 1 case due to aneurysm formation. With respect to the pathological changes of the artery in those 6 cases of ill prognosis, 1 case of haemorrhagic death had the

Table 1. Progress, pathological and biochemical findings in the operation of synthetic blood-vessel grafting for pulseless disease reported in the literatures in our country.
cm. cd. a. : common carotid artery ; subcl. a. : subclavian artery ; bc.a. : brachiocephalic artery ; sr. : erythrocyte sedimentation rate.

author	age sex	grafting portions	progress	pathological findings	biochemical findings
SIMIZU, K.	——	——	still opening in 2.5 years	——	——
TAKEUCHI	17 F	bc. a.–r.cm.cd.a.	hemiplegia on the following day of operation. died on the 9th day	abundant cell infiltration over the whole layers of the aorta and cd.a.	——
KATSUNUMA	18 F	bypass	improved	granulation and round cell infiltration in the tunica media	——
KARUBE	20 F	aorta < r.cm.cd.a. / l.cm.cd.a.	died from thrombosis on the 18th day	diffuse productive inflammation of the aorta, l. and r. cd., vertebral and subcl. a.	——
KARUBE	20M	aorta–r.cm.cd.a.	still opening in 9 months	——	——
WAKABAYASHI	21 F	aorta < r.cm.cd.a. / r.subcl.a.	aneurysm formed over the whole anastomosis two years later	severe inflammation of the aorta and cd. a.	CRP strongly positive, marked acceleration of sr.
KOGA	30 F	aorta < r.cd.a. / l.cd.a.	still opening in 9 months	the cicatrization phase. no cell infiltration	mild acceleration of sr.
KOGA	30 F	bc.a.–r.cm.cd.a.	still opening in a year	the cicatrization phase	mild acceleration of sr.
KOGA	29 F	bc.a. < r.cd.a. / l.cd.a.	still opening in 4 months	the cicatrization phase	mild acceleration of sr.
KOGA	40 F	bc.a. < r.cm.cd.a. / l.subcl.a.	still opening in 9 months	the cicatrization phase	mild acceleration of sr.
KIMOTO	32 F	aorta < l.cm.cd.a. / l.subcl.a.	opening on leaving the hospital	——	sr. S_1 40mm
WADA	38 F	aorta < r.cm.cd.a. / r.subcl.a.	opening on leaving the hospital	——	——
INOKUCHI	32 F	aorta < l.cm.cd.a. / l.subcl.a.	died of internal haemorhage into the thoracic cavity and cardiac insufficiency	pathological changes extending as far as the coronary artery and the valve of aorta	——

KUBO	——	aorta-l. subcl. a., endarterectomy in the first portion of the l. cm.cd.a.	still opening in a year	cell infiltration in the tunica interna et media	——
KUBO	——	aorta-l.cm.cd.a.	opening	——	——
KUBO	——	bypass	died from cardiac tamponade on the 10th day	——	——
TAKAHASHI	19 F	aorta < r.cd.a. / r.subcl.a. ; aorta-l.cm.cd.a.	died from infection in two years	——	——
NISHINO	42 F	aorta < r.cm.cd.a. / l.cm.cd.a.	still opening in 1.5 years	the cicatrization phase	CRP (−), RAT (−), ASLO 160 TODD u, sr. S_1 35mm
NISHINO	25 F	aorta < l.cm.cd.a. / l.subcl.a.	died from thrombosis on the 10th day	severe acute inflammation of the aorta and carotid artery	CRP & RAT strongly positive. ASLO 333 TODD u, sr. S_1 126mm

changes extending as far as the coronary artery and the aortic valves, and died from internal haemorrhage in the thoracic cavity and cardiac insufficiency after the operation (INOKUCHI). Another case had not been mentioned of its pathologic and biochemical findings (KUBO). In the remaining 4 cases, pathological and biochemical pictures of severe inflammation were obviously mentioned (TAKEUCHI, KARUBE, WAKABAYASHI, NISHINO). Of them, 2 cases had a marked promotion of blood sedimentation, an increased level of ASLO, and the strongly positive CRP and RAT (WAKABAYASHI, NISHINO). On the other hand, there were 13 cases of good prognosis, of which 5 cases were uncertain, but 8 cases were reported to be in a subsiding stage of inflammation.

Although the number of the cases examined was small, comparison was made between the 2 cases we examined. In Case 1, the aortic wall was almost normal, but the pathologically changed brachiocephalic, common carotid and subclavian arteries were almost obstructed in the inner cavities, cicatrized in a cord shape, and had a small amount of lymphocytes in the surrounding of the nutrient vessels in the adventitia, with the clinical pictures of erythrocyte sedimentation being S_1 35 mm, ASLO 160 TODD u., RAT (−) and CRP (−). In Case 2, the thoracic aorta, brachiocephalic and carotid arteries had severe inflammatory hypertrophy, and there was inflammatory cell infiltration of plasmocytes, lymphocytes and acidophils in the tunica media as well as granulation incorporating the LANGHANS' giant cells. Besides, a serious increase of the round cells was seen in the tunica interna and adventitia, with the clinical pictures of erythrocyte sedimentation being S_1 126 mm, CRP and RAT being strongly positive, and ASLO being 333 TODD u. In short, obvious pathological and biochemical differences were observed between the two cases in our examination. Thus, the grafting produced better results than thromboendoarterectomy, but not so excellent in atherosclerosis. This pathophysiological difference influences the prognosis, indicating that the examination concerning the application of the operation viewed from the inflammatory pictures is of great importance in the case

of pulseless disease.

The erythrocyte sedimentation rate in pulseless disease has been examined in almost all the cases, but it is hardly said that ASLO, CRP and RAT have been generally determined. Above all, it was in only one case reported by WAKABAYASHI that was examined from the standpoint of indication for surgical operation. On literature[27)29)36)37)38)45)54)55)58)], CRP and RAT were positive in almost all the cases which reported (Table 2), but negative in 1 case of SATOMI and 1 case of ours. As for the ASLO value, its increase was seen in 4 out of 10 cases. NAGATA, WAKISAKA, SATOMI, WAKABAYASHI, KIMURA, KATO and others stated that these laboratory findings on pulseless disease could be normalized by administration of adrenocortical hormone. However, SATOMI reported that no improvement could be obtained in 2 out of his 4 cases. Similarly in our Case 2, the promoted erythrocyte sedimentation was fairly improved (from 120 mm to 40 mm in the S_1 value), but CRP remained strongly positive. On the basis of his experience with operation, WAKABAYASHI stated that the pathological changes of the blood vessels still resist in the case with the strongly positive finding in the CRP even after CRP had been apparently turned negative by internal treatment, and accordingly that careful attention should be paid at operation. As pointed out by ABURAI[1)], NASU[39)] and KAMITANI[24)], there are

Table 2. Erythrocyte sedimentation rate, CRP, RAT, ASLO and efficacy of adrenocortical hormone in pulseless disease reported in the literatures.

author	erythrocyte sedimentation rate (mm)	CRP	RAT	ASLO (TODD u)	effect of adrenocortical hormone
1. KATO	accelerated	++	—	—	both erythrocyte sedimentation and CRP improved
2. KIMURA	accelerated	+	—	—	both erythrocyte sedimentation and CRP improved
3. NAKAGAWA	accelerated	++	+	—	—
4. NAGATA K.	—	+++	—	50	CRP improved and negative
5. NAGATA Y. (several cases)	accelerated	+	—	—	—
6. NISHINO	35	−	−	160	—
7. NISHINO	126	+++	+++	333	only erythrocyte sedimentation improved
8. SATOMI (4 cases)	accelerated	+++ (3 cases)	++ (3 cases)	increased (1 case)	2 cases improved
9. WAKABAYASHI	strongly accelerated	++	—	—	improved
10. WAKISAKA	strongly accelerated	+++	+++	833	improved
11. WAKISAKA	mild accelerated	+	++	12	improved

pathological transitions from the acute to the cicatrization phases in pulseless disease, and therefore it may be possible to transfer the condition of this disease into the cicatrization phase as soon as possible by internal treatment and then to perform in the earlier time the surgical treatment for improvement of cerebral circulation in the case of pulseless disease, excluding such cases as have a rapid progress of the condition and irreversible disturbances of the cerebral circulation. In any way, our statistical examination showed that the grafting performed in the stage showing an acute inflammatory state might cause grave prognosis. In order to discriminate such cases prior to the operation, it is necessary to examin the erythrocyte sedimentation, CRP, RAT and ASLO as a mean of the screening test, and additionally to pay attention to the points mentioned by WAKABAYASHI.

In recent years, the problem of preventing the cerebral circulatory disturbances due to the blood stream blockage at the time of operation for reconstruction of the carotid artery is being solved to a fairly large extent. As to the time of blood-stream blockage in the carotid artery, there are several reports on successful operations without leaving any cerebral circulatory disturbances carried out by blocking for 20 minutes under low body temperature (COOLEY, 1956[7]), for 17 minutes under normal temperature (ROB, 1957[43]), for more than 1 hour under low body temperature (WEGNER, 1958[56]), for 5 to 30 minutes (DE BAKEY, 1959[9]), for 20 minutes under normal temperature (WADA, 1961[53]), for 10 to 50 minutes (INOKUCHI, 1961[19]), for 25 to 60 minutes under normal temperature with the head on an ice pillow (KOGA, 1963[31]), for 25 minutes under nomal temperature (ISHIGAMI, 1964[20]), for 60 to 95 minutes under low body temperature of 29 to 30°C with the head on an ice pillow (TAKAHASHI, 1965[47]), and for 40 minutes under low body temperature of 31.4°C (KAGEYAMA, 1965[22]). Generally, the possibility of blocking the carotid artery during the operation is decided by whether or not the compression of the internal carotid artery before the operation might cause disturbances of consciousness, fits of convulsion, and other nervous disturbances (MATAS' test). On the other hand, repeated compressions of the carotid artery were tried in order to prolong its tolerance time. Such a trial seems, likewise, unable to produce satisfactory results in the cases of bilateral carotid stenosis complicated by obstruction and circulatory insuffciency of the vertebral artery or circulus arteriosus Willisi. It is necessary to maintain the cerebral circulation during the blockage by the external or intraluminal shunt (COOLEY, 1956[7] : DE BAKEY, 1959[9]). DE BAKEY and FREEMANN et al.[13] stated that local anesthesia was more advantageous in the cases requiring no thoracotomy than the general one because the former allowed to observe the condition of a patient's consciousness, and that it might be hardly advisable to utilize the low body temperature method with an attempt of preventing the cerebral circulatory disturbances because it might promote a decrease in the amount of blood circulation in the brain. For the premedication, such cerebral depressants as opiates and barbiturates had better not be used, or if any, used as little as possible. Against the reflex of the glomus caroticum, they advocated to prevent hypotension by administration of atropine and Neosynephrine when the reflex still remained even after blocking it by procaine infiltration into the surrounding, and to maintain the peripheral blood pressure at a level of 200 mm.Hg using Epinephrine during the blockage. With the local anesthesia alone, how-

ever, there are actually some difficulties for the patients who are not cooperative or in semicoma ; additionally, there is rather a danger of causing cerebral hypoxia if the patient is in unrest and fails to have sufficient respiration.

WELLS et al. (1963)[57] used an electroencephalogram as a monitor for the examination of the state of cerebral circulation. He reported that slow (delta) waves appeared 15 to 30 seconds after the compression, followed by tonic and clonic convulsions on the other side, in 4 out of the 19 cases which were examined by the carotid arterial compression test before the operation. The blocking of long duration (16 minutes) under general anesthesia did not leave any nervous disturbances in those patients. In other 4 cases, the delta waves appeared within 30 seconds, causing unrest, syncope, asthenia and blacking out ; but the delta waves disappeared and those symptoms were improved while the compression was being given (60 to 90 seconds). These signify that if these were not due to the increased tolerance of the cerebral tissues against ischemia, the increased blood supply through the collateral routes might be obtainable more than expected. In the remaining 11 cases, neither electroencephalographic abnormalities nor nervous symptoms were caused. Thereupon, he considered general anesthesia with oxygen-rich gas mixtures, mild hypercarbia (by adding 5 % CO_2 to the oxygen) and induced systemic hypertension (blood pressure of more than 160 mmHg) as being the best for prevention of cerebral circulatory disturbances at blocking of the cartoid artery, and obtained success in 55 cases except for the one in which anesthesia was given unskilfully among 56 cases treated with the blocking for 5 to 30 minutes.

While WELLS used electroencephalography as the monitor, LYONS et al. (1964)[33] considered that electroencephalography could not be a sufficiently reliable monitor in the patients with obstruction of the vertebral artery, lack of the posterior communicating artery or with incomplete circulation in these arteries ; and then on the basis of the experimental fact that neurological disturbances appear when the level of PO_2 in the cerebral venous blood falls below 50 % he advocated, as a monitor enabling the blocking during the operation, to determine the PO_2 amount of the cerebral venous blood after inserting a catheter into the internal jugular vein as far as the lateral sinus. It seems that the blocking of the ramification to the peripheral side only matters in such patients as have complete obstruction of the common carotid arteries bilaterally but the vascular opening in the area from the ramification to the peripheral sides like our Case 1, which required the blocking for 15 minutes on the right side and for 30 minutes on the left side. In Case 2, the blocking of the innominate artery was performed for 57 minutes and of the left carotid artery for 15 minutes under normal temperature. Both the cases suffered from no neurological sequela.

With respect to the postoperative anticoagulant therapy, KOGA[31] administered continuously by intravenous dripping, of Heparin in dosage of 150 to 200 mg daily for 4 to 6 days following the artificial vascular transplantation in 4 cases of pulseless disease, and found no thrombosis in any of his cases. FOGARTY et al. (1965)[12] reported that they administered Heparin maintaining double the LEE-WHITE clotting time for the initial 72 hours and then 3 times of the same for the following 7 days, and subsequently employed prothrombin depressants for a long term. In our Case 1, Heparin was administered in

dosage of 150 mg daily by continuous drip injection for 7 days immediately after the operation, and at the same time the administration of Streptokinase was combined. Since the 7 th postoperative day, Nicoumalone and Chymotrypsin were simultaneously administered for 1 month. Case 2 was treated with Heparin administered in dosage of 100 to 150 mg daily by continuous drip injection for 9 days immediately after the operation, but suffered from thrombosis on the 9th day of the treatment. In other words, it was shown that pulseless disease, as mentioned above, had vascular changes of the acute phase which could not be controlled by the anticoagulant agent alone.

It is commonly admitted that histologically, the main pathological changes in this disease are panvasculitis in the tunica media and in the transitional area from the tunica media to the adventitia, the pathological changes of the tunica interna and thrombosis is the secondary one, and this disease can be distiguished from BURGER's disease by the lack of fibrinoid degeneration[32)39)46)]. ABURAI observed the pathological changes corresponding to the cicatrization phase following the acute phase in his autopsy findings. NASU (1959)[39)] histologically classified his 18 autopsied cases under two broad categories of granulomatous changes and diffuse productive inflammation, and made a further classification of the pathologic pictures as follows:

1) Granulomatous changes
   a) Coagulation necrosis
   b) Tuberculoid type with Langhans' giant cells
2) Diffuse productive inflammation
   a) Type mainly with lymphocyte and plasmocyte infiltration incorporating giant cells
   b) Cicatrized obstructive arterial sclerostenosis

In addition, KAMITANI[24)] made the following classification:

Type I Inflammation of the adventitia
Type II Nodal granuloma
Type III Giant-cell arteritis
Type IV Diffuse productive inflammation

In his explanation, Type I seems to enter the cicatrization phase, with vasculitis being localized mainly in the adventitia, and the muscle fibers of the tunica media have disappeared while there remain the fragmented elastic fibers. No mention was given on the cicatrization phase of the other 3 types. In Case 1, we examined in the present study, complete disappearance of the elastic and muscle fibers from the tunica media and fibrosis of both the tunica media and adventitia led the condition to the cicatrization phase. Presumably, it might have shifted from what KAMITANI called Type II or Type III into the cicatrization phase, and on the other hand, it may be considered as belonging to the type of cicatrized obstrutive arterial sclerostenosis according to NASU's classification. Case 2 consisted mainly of serious inflammation with lymphocyte, acidophil and plasmocyte infiltration incorporating giant cells in the tunica media and the transitional area from the tunica media to the adventitia; and corresponds to KAMITANI's Type III or NASU's type of "diffuse productive inflammation mainly with lymphocyte and plasmocyte infiltration incorporating giant cells". In any way, there is a cicatrization phase following an acute

inflammatory phase in this disease. Therefore, how we can transfer the condition from its acute phase into the cicatrization phase causing as few sequela as possible is the primary thema of the surgical treatment for reconstruction of blood vessels as well as the thema in the internal treatment of this disease.

CONCLUSIONS

Two cases of pulseless disease in which the blood circulation between the ascending aorta and the peripheral carotid arteries had been surgically reconstructed using a Y-shaped tetoron synthetic blood vessel were reported in detail on their clinical courses, laboratory findings, operations and histological findings. In addition, the prognosis of this disease was examined on the basis of the biochemical and pathological findings in the cases of synthetic vascular transplantation that had been reported in this country as well as in our 2 cases. The results of the operation were poor in this disease as compared with the cases of synthetic vascular transplantation performed against vascular obstruction due to atherosclerosis. This was because of the inflammatory states and of the pathologic histological difference that this disease had its main pathological changes of panvasculitis in the tunica media and the transitional area from the tunica media to the adventitia. The laboratory examinations of blood sedimentation, ASLO, CRP and RAT clarified such states of inflammation and were helpful as screening in application of the operation. On the other hand, administration of adrenocortical hormone could normalize those laboratory findings to a fairly large extent, but there often remained the pathologicall serious changes. It is, therefore, necessary to pay further careful attention at operation. Besides, the permissible time of blocking the carotid artery at operation was also examined on the recently available literatures.

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和文抄録

脈なし病の外科的治療

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Y型 Tetoron 代用血管を用いて上行大動脈と頭側頸動脈との間に血行を再開した脈なし病の2病例について，その臨床経過，諸検査成績，手術および組織所見などについて詳述した．そして本邦における脈なし病に対する代用血管移植症例にわれわれの2例を加え，その予後を生化学的，病理学的所見より検討して，atherosclerosis による血管閉塞に対する代用血管移植術の場合に比べて，その手術成績が悪いのは炎症状態と本症が中膜および中膜と外膜との移行部に主病変をおく汎血管炎であるという病理組織学的差異に原因があることを述べた．赤沈，ASLO, CRP, RAT などの諸検査はこの炎症状態を知り，手術適用の Screening として有用である．一方副腎皮質ホルモンの投与によつてこれらの諸検査成績は可成り正常化できるが，尚病理学的に重篤な変化が残ることがあるので更に手術に際しては十分の注意をはらうべきである．又手術時頸動脈遮断時間についても最近の文献的考察を合わせ行なつた．